

Troponin T in COVID-19 hospitalized patients: kinetics matter

M.L. Luchian¹, A.I. Motoc¹, S. Lochy¹, J. Magne², B. Roosens¹, D. Belsack¹, K. Van Den Bussche¹, B. Von Kemp¹, X. Galloo¹, C.E. Francois¹, L. Seyler¹, J. Van Laethem¹, C. Weytjens¹, S. Droogmans¹, B. Cosyns¹

¹University Hospital (UZ) Brussels, Brussels, Belgium; ²Dupuytren University Hospital Centre Limoges, Limoges, France

Funding Acknowledgement: Type of funding sources: None.

Background: Coronavirus disease 2019 (COVID-19) pandemic continues to overwhelm healthcare systems worldwide, due to high numbers of critical cases over a short period of time (1,2). Elevated cardiac troponin (cTn), suggestive for myocardial damage, was associated with increased mortality of COVID-19 patients (3,4). However, data addressing the role of cTn in major adverse cardiovascular events (MACE) in COVID-19 patients is scarce.

Objectives: We aimed to assess the role of baseline cTnT and cTnT kinetics in the prediction of MACE and in-hospital mortality in COVID-19 patients. Furthermore, we assessed the association between cTnT kinetics and the need of cardiac imaging evaluation.

Methods: 310 patients were included prospectively (age 64.6±16.7 years, 180 (58.1%) males), between March 2020 and April 2020. Clinical data including demographics, medical history, comorbidities, clinical evaluation, laboratory exams, in-hospital treatment, complications and outcomes were collected at admission and during hospitalization by physicians in charge. Two hundred and two patients (65.1%) with at least two cTnT values assessed during hospitalization, at 24–48 hours interval were included in the final analysis. cTnT-values >0.011 micrograms/L were considered elevated, according to hospital laboratory cut-offs. Patients were divided into 3 groups according to cTnT kinetics profile: 1 – variable, 2 – descending and 3 – constant. cTnT slope was defined as the ratio of the cTnT change and the change in time. MACE were considered as the primary

endpoint and were composed by all-cause mortality, acute heart failure, acute coronary syndrome, pericarditis, myocarditis, atrial fibrillation or flutter and pulmonary embolism. In-hospital mortality was considered as the secondary endpoint.

Results: Mean hospitalization was 13.9±0.9 days. MACE occurred in 60 patients (29.7%) and in-hospital mortality in 40 (19.8%) patients. Baseline cTnT independently predicted MACE, (p=0.047, HR 1.805, 95% CI 1.009–3.231) and in-hospital mortality (p=0.009, HR 2.322, 95% CI 1.234–4.369) (Figure 1A, 1B). An increased cTnT slope independently predicted in-hospital mortality (p=0.041, HR 1.006, 95% CI 1.000–1.011). Constant cTnT was associated with lower MACE and mortality rates (p=0.000, HR 3.080, 95% CI, 1.914–4.954, p=0.000, HR 2.851, 95% CI 1.828–4.447, respectively) (Figure 1C, 1D, 2). Cardiac imaging evaluation was performed in 8 (16%) patients with constant cTnT, 30 (60%) with variable cTnT, and 12 (24%) with descending cTnT. (p<0.001)

Conclusions: Increased baseline cTnT independently predicted MACE and in-hospital mortality in COVID-19 patients. The magnitude of cTnT increase over time was associated with in-hospital mortality. On the contrary, patients with constant cTnT had lower MACE and in-hospital mortality rates. These findings emphasize the additional role of cTnT testing in COVID-19 patients for risk stratification and improved diagnostic pathway and management

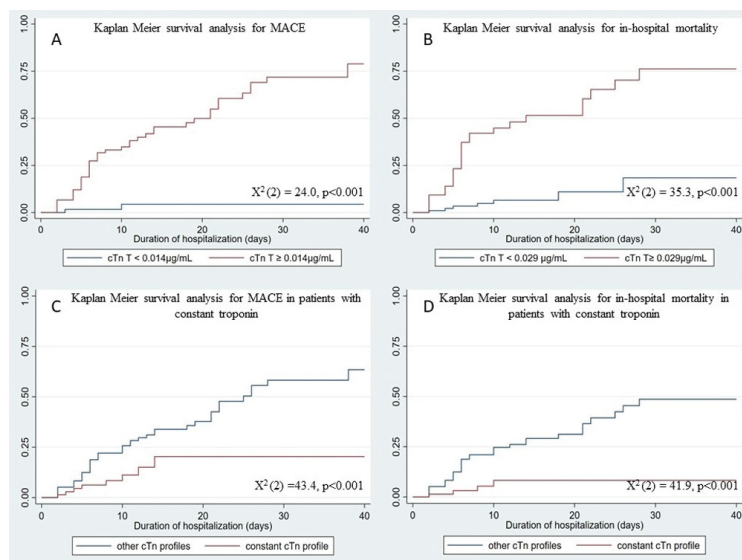


Figure 1. Kaplan Meier for MACE and mortality

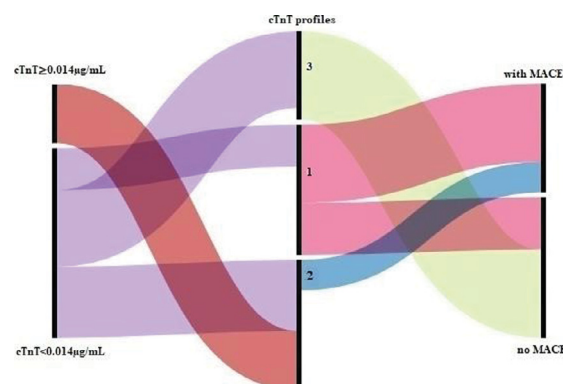


Figure 2. Troponin kinetics as MACE predictors